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NEWS	15	MAR 31	CAS REGISTRY enhanced with additional experimental spectra
NEWS	16	MAR 31	CA/CAplus and CASREACT patent number format for U.S. applications updated
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NEWS	18	MAR 31	EMBASE, EMBAL, and LEMBASE reloaded with enhancements
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NEWS	20	APR 15	WPIDS, WPINDEX, and WPIX enhanced with new predefined hit display formats

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FILE 'HOME' ENTERED AT 12:48:42 ON 17 APR 2008

=> file medline caplus embase biotechno biosis scisearch
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   2.73       2.73

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=> s ires
L1        9926 IRES

=> s l1 and hcv
L2        1508 L1 AND HCV

=> s l1 and hepatitis
L3        2110 L1 AND HEPATITIS

=> s hcv ires
L4         907 HCV IRES

=> s l2 and inhibitor
L5         170 L2 AND INHIBITOR

=> s l3 and inhibitor
L6         211 L3 AND INHIBITOR

=> s l6 not l5
L7          46 L6 NOT L5

=> d ti 1-46

L7   ANSWER 1 OF 46     MEDLINE on STN
TI   A search for structurally similar cellular internal ribosome entry sites.

L7   ANSWER 2 OF 46     MEDLINE on STN
TI   Zuotin, a DnaJ molecular chaperone, stimulates cap-independent translation
     in yeast.

L7   ANSWER 3 OF 46     MEDLINE on STN
TI   Genome-wide RNAi screen reveals a specific sensitivity of IRES
     -containing RNA viruses to host translation inhibition.

L7   ANSWER 4 OF 46     MEDLINE on STN
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TI BCL-2 translation is mediated via internal ribosome entry during cell stress.

L7 ANSWER 5 OF 46 MEDLINE on STN
TI Translation of cellular inhibitor of apoptosis protein 1 (c-IAP1) mRNA is IRES mediated and regulated during cell stress.

L7 ANSWER 6 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Method for quantifying expression of a target protein candidate in a cell identifying a target protein of a small molecule modulator for use in drug screening applications

L7 ANSWER 7 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI 2'-C-Methyl-Ribofuranosyl Cytidine Prodrugs, Pharmaceutical Compositions and Uses Thereof

L7 ANSWER 8 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI A search for structurally similar cellular internal ribosome entry sites

L7 ANSWER 9 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Zuotin, a DnaJ molecular chaperone, stimulates cap-independent translation in yeast

L7 ANSWER 10 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Method of screening for inhibitors of IRES-mediated translation and identification of antiviral peptides targeted to hepatitis C virus

L7 ANSWER 11 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Oligonucleotide microarrays comprising nucleic acid analogs for hybridization with target RNA, including RNA in nucleoprotein complexes

L7 ANSWER 12 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Expression of a recombinant protein from a complementary transgene sequence in a plant cell using a positive strand ssRNA virus replication, and method of conferring disease resistance to a transgenic plant

L7 ANSWER 13 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Virally encoded RNAs as substrates, inhibitors, and delivery vehicles for RNAi and uses for antiviral therapy

L7 ANSWER 14 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Genome-wide RNAi screen reveals a specific sensitivity of IRES -containing RNA viruses to host translation inhibition

L7 ANSWER 15 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI BCL-2 Translation Is Mediated via Internal Ribosome Entry during Cell Stress

L7 ANSWER 16 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Translation of cellular inhibitor of apoptosis protein 1 (c-IAP1) mRNA is IRES mediated and regulated during cell stress

L7 ANSWER 17 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Methods for identifying small molecules that bind specific RNA structural motifs and uses high-throughput screening of combinatorial librarys

L7 ANSWER 18 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Variants of the p40 subunit of interleukin 12 with altered glycosidation for use as adjuvants in vector vaccines

L7 ANSWER 19 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN

TI Replicon-based activation of endogenous genes using genetic elements for RNA replication

L7 ANSWER 20 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Gene transfer vectors for treating autoimmune diseases and diseases with immunopathogenesis

L7 ANSWER 21 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Hepatitis C inhibitor peptides

L7 ANSWER 22 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Interference with viral ires-mediated translation by a small yeast rna reveals critical rna-protein interactions

L7 ANSWER 23 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI RNA virus vector and helper virus or cell line for gene cloning, vaccine development, and neoplasm and inflammation inhibitor recombinant production

L7 ANSWER 24 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI RNA oligonucleotide inhibitor of protein formation initiation factor binding by mRNA internal ribosome entry site, mRNA of virus, and oligonucleotide use in animal viral infection treatment

L7 ANSWER 25 OF 46 CAPLUS COPYRIGHT 2008 ACS on STN
TI Methods for screening compounds for potential inhibitors of translation of viral RNAs for use as antiviral agents

L7 ANSWER 26 OF 46 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
TI A search for structurally similar cellular internal ribosome entry sites.

L7 ANSWER 27 OF 46 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
TI Zuotin, a DnaJ molecular chaperone, stimulates cap-independent translation in yeast.

L7 ANSWER 28 OF 46 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
TI Genome-wide RNAi screen reveals a specific sensitivity of IRES -containing RNA viruses to host translation inhibition.

L7 ANSWER 29 OF 46 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
TI BCL-2 translation is mediated via internal ribosome entry during cell stress.

L7 ANSWER 30 OF 46 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
TI Translation of cellular inhibitor of apoptosis protein 1 (c-IAP1) mRNA is IRES mediated and regulated during cell stress.

L7 ANSWER 31 OF 46 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI A search for structurally similar cellular internal ribosome entry sites.

L7 ANSWER 32 OF 46 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Zuotin, a DnaJ molecular chaperone, stimulates cap-independent translation in yeast.

L7 ANSWER 33 OF 46 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on

STN
TI Ribosomal protein L13a inhibits translation by blocking the formation of 80S complex on the GAIT element containing mRNA: Dependence on the translation initiation factor eIF4G.

L7 ANSWER 34 OF 46 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Genome-wide RNAi screen reveals a specific sensitivity of IRES -containing RNA viruses to host translation inhibition.

L7 ANSWER 35 OF 46 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI BCL-2 translation is mediated via internal ribosome entry during cell stress.

L7 ANSWER 36 OF 46 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Inhibitors of protein synthesis identified by a high throughput multiplexed translation screen.

L7 ANSWER 37 OF 46 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Translation of cellular inhibitor of apoptosis protein 1 (c-IAP1) mRNA is IRES mediated and regulated during cell stress.

L7 ANSWER 38 OF 46 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI A search for structurally similar cellular internal ribosome entry sites

L7 ANSWER 39 OF 46 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Incorporation of an internal ribosome entry site-dependent mechanism in arsenic-induced GADD45 alpha expression

L7 ANSWER 40 OF 46 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Zuotin, a DnaJ molecular chaperone, stimulates cap-independent translation in yeast

L7 ANSWER 41 OF 46 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Two internal ribosome entry sites mediate the translation of p53 isoforms

L7 ANSWER 42 OF 46 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI A B-12-responsive internal ribosome entry site (IRES) element in human methionine synthase

L7 ANSWER 43 OF 46 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Genome-wide RNAi screen reveals a specific sensitivity of IRES -containing RNA viruses to host translation inhibition

L7 ANSWER 44 OF 46 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI BCL-2 translation is mediated via internal ribosome entry during cell stress

L7 ANSWER 45 OF 46 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Translation of cellular inhibitor of apoptosis protein 1 (c-IAP1) mRNA is IRES mediated and regulated during cell stress

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L7   ANSWER 46 OF 46  SCISEARCH  COPYRIGHT (c) 2008 The Thomson Corporation  on
     STN
TI   Functional interactions in internal translation initiation directed by
     viral and cellular IRES elements


=> d his

     (FILE 'HOME' ENTERED AT 12:48:42 ON 17 APR 2008)

     FILE 'MEDLINE, CAPLUS, EMBASE, BIOTECHNO, BIOSIS, SCISEARCH' ENTERED AT
     12:56:22 ON 17 APR 2008
L1          9926 S IRES
L2          1508 S L1 AND HCV
L3          2110 S L1 AND HEPATITIS
L4           907 S HCV IRES
L5           170 S L2 AND INHIBITOR
L6           211 S L3 AND INHIBITOR
L7            46 S L6 NOT L5

=> dup rem l5
PROCESSING COMPLETED FOR L5
L8            71 DUP REM L5 (99 DUPLICATES REMOVED)

=> s l4 and inhibitor
L9          108 L4 AND INHIBITOR

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10           31 DUP REM L9 (77 DUPLICATES REMOVED)

=> d ti 1-31

L10  ANSWER 1 OF 31     MEDLINE on STN                   DUPLICATE 1
TI   The picornavirus avian encephalomyelitis virus possesses a hepatitis C
     virus-like internal ribosome entry site element.

L10  ANSWER 2 OF 31  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Combinations comprising HCV protease inhibitor(s) and
     HCV IRES inhibitor(s)

L10  ANSWER 3 OF 31     MEDLINE on STN                   DUPLICATE 2
TI   Inhibition of hepatitis C virus internal ribosome entry site-mediated
     translation by an RNA targeting the conserved IIIf domain.

L10  ANSWER 4 OF 31  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Establishment and evaluation of a cell-based drug screening model used for
     screening HCV-IRES inhibitor

L10  ANSWER 5 OF 31  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Preparation of indoles for prevention or treatment of Hepatitis C virus
     (HCV) infection.

L10  ANSWER 6 OF 31  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Methods for producing hepatitis C virus ARFP/F/Core+1 protein and its use
     in screening for antiviral agents

L10  ANSWER 7 OF 31     MEDLINE on STN                   DUPLICATE 3
TI   Initiation of protein synthesis by hepatitis C virus is refractory to
     reduced eIF2.GTP.Met-tRNA(i)(Met) ternary complex availability.
```

L10 ANSWER 8 OF 31 MEDLINE on STN DUPLICATE 4
TI A peptide derived from RNA recognition motif 2 of human la protein binds to hepatitis C virus internal ribosome entry site, prevents ribosomal assembly, and inhibits internal initiation of translation.

L10 ANSWER 9 OF 31 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Interfering with hepatitis C virus IRES activity using RNA molecules identified by a novel in vitro selection method

L10 ANSWER 10 OF 31 MEDLINE on STN DUPLICATE 5
TI Enhancement of internal ribosome entry site-mediated translation and replication of hepatitis C virus by PD98059.

L10 ANSWER 11 OF 31 MEDLINE on STN DUPLICATE 6
TI Differential effects on the hepatitis C virus (HCV) internal ribosome entry site by vitamin B12 and the HCV core protein.

L10 ANSWER 12 OF 31 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 7
TI Biaryl guanidine inhibitors of in vitro HCV-IRES activity

L10 ANSWER 13 OF 31 MEDLINE on STN DUPLICATE 8
TI Virological effects of ISIS 14803, an antisense oligonucleotide inhibitor of hepatitis C virus (HCV) internal ribosome entry site (IRES), on HCV IRES in chronic hepatitis C patients and examination of the potential role of primary and secondary HCV resistance in the outcome of treatment.

L10 ANSWER 14 OF 31 MEDLINE on STN DUPLICATE 9
TI Riboproteomics of the hepatitis C virus internal ribosomal entry site.

L10 ANSWER 15 OF 31 MEDLINE on STN DUPLICATE 10
TI Demonstrating internal ribosome entry sites in eukaryotic mRNAs using stringent RNA test procedures.

L10 ANSWER 16 OF 31 MEDLINE on STN DUPLICATE 11
TI Inhibitor RNA blocks the protein translation mediated by hepatitis C virus internal ribosome entry site in vivo.

L10 ANSWER 17 OF 31 MEDLINE on STN DUPLICATE 12
TI Molecular dynamics simulation of hepatitis C virus IRES IIId domain: structural behavior, electrostatic and energetic analysis.

L10 ANSWER 18 OF 31 CAPLUS COPYRIGHT 2008 ACS on STN
TI Methods for identifying polypeptide factors interacting with RNA

L10 ANSWER 19 OF 31 MEDLINE on STN DUPLICATE 13
TI Characterization of the expression of the hepatitis C virus F protein.

L10 ANSWER 20 OF 31 MEDLINE on STN DUPLICATE 14
TI A small yeast RNA inhibits HCV IRES mediated translation and inhibits replication of poliovirus in vivo.

L10 ANSWER 21 OF 31 MEDLINE on STN DUPLICATE 15
TI A potent and specific morpholino antisense inhibitor of hepatitis C translation in mice.

L10 ANSWER 22 OF 31 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 16
TI Inhibitory effect of IRES specific inhibitor RNA on HCV IRES mediated protein translation

L10 ANSWER 23 OF 31 MEDLINE on STN
TI Determinants of hepatitis C translational initiation in vitro, in cultured cells and mice.

L10 ANSWER 24 OF 31 MEDLINE on STN DUPLICATE 17
TI Effect of inhibitor RNA on intracellular inhibition of viral gene expression in 5'-noncoding region of hepatitis C virus.

L10 ANSWER 25 OF 31 MEDLINE on STN DUPLICATE 18
TI In vitro cleavage of eIF4GI but not eIF4GII by HIV-1 protease and its effects on translation in the rabbit reticulocyte lysate system.

L10 ANSWER 26 OF 31 MEDLINE on STN DUPLICATE 19
TI Vitamin B12 stalls the 80 S ribosomal complex on the hepatitis C internal ribosome entry site.

L10 ANSWER 27 OF 31 MEDLINE on STN DUPLICATE 20
TI Involvement of proteasome alpha-subunit PSMA7 in hepatitis C virus internal ribosome entry site-mediated translation.

L10 ANSWER 28 OF 31 MEDLINE on STN DUPLICATE 21
TI Hepatitis C IRES: translating translation into a therapeutic target.

L10 ANSWER 29 OF 31 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Hepatitis C viral IRES inhibition by phenazine and phenazine-like molecules.

L10 ANSWER 30 OF 31 MEDLINE on STN
TI Inhibition of internal entry site (IRES)-mediated translation by a small yeast RNA: a novel strategy to block hepatitis C virus protein synthesis.

L10 ANSWER 31 OF 31 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Inhibition of internal entry site (IRES)-mediated translation by a small yeast RNA: A novel strategy to block hepatitis C virus protein synthesis.

=> d ab 16 13 20 22

L10 ANSWER 16 OF 31 MEDLINE on STN DUPLICATE 11
AB AIM: To investigate the inhibitory effect of hepatitis C virus internal ribosome entry site (HCV IRES) specific inhibitor RNA (IRNA) on gene expression mediated by HCV IRES in vivo. METHODS: By using G418 screening system, hepatoma cells constitutively expressing IRNA or mutant IRNA (mIRNA) were established and characterized, and HCV replicons containing the 5' untranslated region (5'UTR) were constructed by using the same method. Cotransfection of pCMVNCRluc containing HCV 5'UTR-luc fusion genes and eukaryotic vector of IRNA into human hepatic carcinoma cells (HepG2) was performed and the eukaryotic expression plasmid of IRNA was transfected transiently into HCV replicons. pCMVNCRluc or pCDNA-luc was cotransfected with pSV40-beta Gal into IRNA expressing hepatoma cells by using lipofectamine 2000 in vitro. Then the reporting gene expression level was examined at 48 h after transfection by using a luminometer and the expressing level of HCV C antigen was analysed with a confocal microscope. RESULTS: Transient expression of IRES specific IRNA could significantly inhibit the expression of reporter gene and viral antigen mediated by HCV IRES by 50% to 90% in vivo, but mIRNA lost its inhibitory activity completely. The luciferase gene expression mediated by HCV IRES was blocked in the HHCC constitutively expressing IRNA. At 48 h after transfection, the expression level of

reporter gene decreased by 20%, but cap-dependent luciferase gene expression was not affected. IRNA could inhibit the HCV replicon expression 24 h after transfection and the highest inhibitory activity was 80% by 72 h, and the inhibitory activity was not increased until 7d after transfection. CONCLUSION: IRNA can inhibit HCV IRES mediated gene expression in vivo.

L10 ANSWER 13 OF 31 MEDLINE on STN DUPLICATE 8
AB Antisense oligonucleotides represent a promising class of antiviral agents. ISIS 14803 is a 20-unit phosphorothioate oligodeoxynucleotide that inhibited hepatitis C virus (HCV) replication and protein expression in cell culture and mouse models. A Phase I dose-escalation clinical study of ISIS 14803 was performed in 24 patients with HCV genotype 1 chronic hepatitis C. The patients received 0.5, 1.0, 2.0 or 3.0 mg/kg of ISIS 14803 for 4 weeks. Two of them receiving 2.0 mg/kg, experienced a significant (>1.0 log10) viral load reduction and nine other patients experienced minor (<1.0 log10) viral load reductions that were difficult to definitively distinguish from assay or patient variations. The aims of this study were to examine the effect of ISIS 14803 on its target site and neighbouring region quasispecies evolution, and to determine whether primary and secondary HCV resistance contributed to the observed virological response rate. The HCV internal ribosome entry site (IRES), including the ISIS 14803 target site in virus specimens collected from patients at baseline and end-of-treatment, was sequenced. An extensive IRES quasispecies analysis was performed in 10 of the patients at various time points before, during and after ISIS 14803 treatment. A significant IRES genetic evolution was found in three out of 10 patients through quasispecies analysis suggesting that treatment with ISIS 14803, a drug designed to bind to HCV RNA, exerted a selective pressure on HCV IRES. However, no mutations in the ISIS 14803 target site, which would inhibit binding of the oligonucleotide to HCV RNA, were detected before (primary resistance) or after treatment (secondary resistance) with the oligonucleotide. Furthermore, no obvious nucleotide changes in the surrounding IRES region that might possibly affect oligonucleotide binding were detected.

L10 ANSWER 20 OF 31 MEDLINE on STN DUPLICATE 14
AB AIM: To investigate the anti-virus infection activity of internal ribosome entry site (IRES) specific inhibitor RNA (IRNA). METHODS: IRNA eukaryotic vector pcRz-IRNA or mIRNA eukaryotic vector pcRz-mIRNA was transfected into human hepatocarcinoma cells (HHCC), then selected with neomycin G418 for 4 to 8 weeks, and then infected with polio virus vaccines line. The cytopethogenesis effect was investigated and the cell extract was collected. At last the polio virus titer of different cells was determined by plaque assay. RESULTS: Constructive expression of IRNA was not detrimental to cell growth. HCV IRES-mediated cap-independent translation was markedly inhibited in cells constructively expressing IRNA compared to control hepatoma cells. However, cap-dependent translation was not significantly affected in these cell line. Additionally, HHCC cells constitutively expressing IRNA became refractory to infection of polio virus. CONCLUSION: IRES specific IRNA can inhibit HCV IRES mediated translation and poliovirus replication.

L10 ANSWER 22 OF 31 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 16
AB Human hepatic carcinoma cell (HHCC) line was transfected with the eukaryotic vectors of internal ribosome entry site (IRES) specific inhibitor RNA (IRNA) or mutant IRNA (pcRz-IRNA or pcRz-mIRNA), and then selected with G418 for 4 wk. HHCC expressing IRNA or mIRNA was cotransfected with pCMVNCRluc containing HCV IRES. HHCC stably expressing pcHCVcluc was transfected with pcRz-IRNA, and pcRz-mIRNA, resp. The luciferase activity was examined at desired time

post-transfection. The pCMVNCRluc was efficiently suppressed in HHCC expressing IRNA rather than the cell line expressing mIRNA. The IRES specific IRNA inhibited the expression of HCV IRES mediated luc gene by 20% to 80% in pcHCVcluc expressing cell after transfection. However, no inhibitory effect of the mutant IRNA was observed PcHCVcluc could be expressed successfully in HHCC, and IRNA inhibited HCV IRES mediated gene expression in vivo.

```
=> s gggagggccctctcg
L11          0 GGGAGGGCCCTCTCG

=> s gggagggccctctcg?
L12          0 GGGAGGGCCCTCTCG?

=> s ?gggagggccctctcg?
L13          0 ?GGGAGGGCCCTCTCG?

=> d his

     (FILE 'HOME' ENTERED AT 12:48:42 ON 17 APR 2008)

     FILE 'MEDLINE, CAPLUS, EMBASE, BIOTECHNO, BIOSIS, SCISEARCH' ENTERED AT
     12:56:22 ON 17 APR 2008
L1         9926 S IRES
L2         1508 S L1 AND HCV
L3         2110 S L1 AND HEPATITIS
L4          907 S HCV IRES
L5          170 S L2 AND INHIBITOR
L6          211 S L3 AND INHIBITOR
L7           46 S L6 NOT L5
L8           71 DUP REM L5 (99 DUPLICATES REMOVED)
L9          108 S L4 AND INHIBITOR
L10          31 DUP REM L9 (77 DUPLICATES REMOVED)
L11           0 S GGGAGGGCCCTCTCG
L12           0 S GGGAGGGCCCTCTCG?
L13           0 S ?GGGAGGGCCCTCTCG?

=> s l2 and sl
L14         30 L2 AND SL

=> s l3 and sl
L15         30 L3 AND SL

=> s l14 or l15
L16         30 L14 OR L15

=> dup rem l16
PROCESSING COMPLETED FOR L16
L17          9 DUP REM L16 (21 DUPLICATES REMOVED)

=> d 1-9 ab

L17  ANSWER 1 OF 9     MEDLINE on STN                    DUPLICATE 1
AB   The minus-IRES ((-)IRES), corresponding to the
     3'-terminal end of the negative strand of hepatitis C virus (
     HCV) RNA, is well conserved among HCV subtypes.  The
     higher order structure of (-)IRES is essential for HCV
     replication, because the viral RNA dependent RNA polymerase, NS5B,
     recognizes it as the initiation site for plus-strand synthesis of the
     HCV genome.  To inhibit the "de novo" synthesis of plus-strand RNA
     molecules, we performed an in vitro selection procedure that is specific
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for the (-)IRES domain I. After confirming the binding convergence in the ninth RNA pool, 42 RNA clones were sequenced and analyzed. Of these, 25 clones (Family-I) had the consensus sequence, 5'-UGGAUC-3', which is complementary to the apical loop of SL -E1, an important region for NS5B recognition. Another 13 clones (Family-II) had the consensus sequence, 5'-GAGUAC-3', which is complementary to the apical loop of SL-D1. Biochemical analyses are in progress to evaluate whether these RNA aptamers have the ability to inhibit HCV replication.

L17 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
AB Disclosed herein is a small peptide, LaR2C, corresponding to the C terminus of RRM2 of the human La protein that binds to the IRES element of hepatitis C virus RNA and its derivs. This invention demonstrates that human La protein interacts with the HCV IRES element both in vitro and in vivo and also shown that this interaction enhances the efficiency of viral RNA translation (Pudi et al, J of Biol Chem, 2003). La protein has three putative RNA recognition motifs (RRM1-3). It has been established that RRM2 binds with high affinity around the GCAC sequence near the initiator AUG and the binding induces a conformational change in the HCV IRES which is critical for the internal initiation of translation (Pudi et al, J of Biol Chem, 2004). TAT-LaR2C fusion protein inhibited HCV IRES mediated translation in pcDNA3-HCV transfected Huh7 cell.

L17 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
AB Translation of the hepatitis C virus (HCV) RNA is mediated by the interaction of ribosomes and cellular proteins with an internal ribosome entry site (IRES) located within the 5' untranslated region (5'UTR). We have investigated whether small RNA mols. corresponding to the different stem-loop (SL) domains of the HCV IRES, when introduced in trans, can bind to the cellular proteins and antagonize their binding to the viral IRES , thereby inhibiting HCV IRES-mediated translation. We have found that an RNA mol. corresponding to SL III of the HCV IRES could efficiently inhibit HCV IRES-mediated translation in a dose-dependent manner without affecting cap-dependent translation. The SL III RNA was also found to bind efficiently to most of the cellular proteins which interacted with the HCV 5'UTR. A smaller RNA corresponding to SL e+f of domain III also strongly and selectively inhibited HCV IRES-mediated translation. This RNA mol. showed strong interaction with the ribosomal S5 protein and prevented the recruitment of the 40S ribosomal subunit by the HCV IRES . In conclusion our results demonstrate a novel approach to selectively block HCV RNA translation using a small RNA mols. mimicking the structure of the stem-loop IIIe+f subdomain of the HCV-IRES. The discovery provides a basis for developing a potent antiviral therapy targeting the interaction between the ribosome and the HCV-IRES RNA.

L17 ANSWER 4 OF 9 MEDLINE on STN DUPLICATE 2
AB Human La protein is known to interact with hepatitis C virus (HCV) internal ribosome entry site (IRES) and stimulate translation. Previously, we demonstrated that mutations within HCV SL IV lead to reduced binding to La-RNA recognition motif 2 (RRM2) and drastically affect HCV IRES -mediated translation. Also, the binding of La protein to SL IV of HCV IRES was shown to impart conformational alterations within the RNA so as to facilitate the formation of functional initiation complex. Here, we report that a synthetic peptide, LaR2C,

derived from the C terminus of La-RRM2 competes with the binding of cellular La protein to the HCV IRES and acts as a dominant negative inhibitor of internal initiation of translation of HCV RNA. The peptide binds to the HCV IRES and inhibits the functional initiation complex formation. An Huh7 cell line constitutively expressing a bicistronic RNA in which both cap-dependent and HCV IRES-mediated translation can be easily assayed has been developed. The addition of purified TAT-LaR2C recombinant polypeptide that allows direct delivery of the peptide into the cells showed reduced expression of HCV IRES activity in this cell line. The study reveals valuable insights into the role of La protein in ribosome assembly at the HCV IRES and also provides the basis for targeting ribosome-HCV IRES interaction to design potent antiviral therapy.

L17 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
AB In our attempt to obtain further information on the replication mechanism of the hepatitis C virus (HCV), we have studied the role of sequences at the 3'-end of HCV minus-strand RNA in the initiation of synthesis of the viral genome by viral RNA-dependent RNA polymerase (RdRp). In this report, we investigated the template and binding properties of mutated and deleted RNA fragments of the 3'-end of the minus-strand HCV RNA in the presence of viral polymerase. These mutants were designed following the newly established secondary structure of this viral RNA fragment. We showed that deletion of the 3'-SL-A1 stem loop significantly reduced the level of RNA synthesis whereas modifications performed in the SL-B1 stem loop increased RNA synthesis. Study of the region encompassing the 341 nucleotides of the 3'-end of the minus-strand RNA shows that these two hairpins play a very limited role in binding to the viral polymerase. On the contrary, deletions of sequences in the 5'-end of this fragment greatly impaired both RNA synthesis and RNA binding. Our results strongly suggest that several domains of the 341 nucleotide region of the minus-strand 3'-end interact with HCV RdRp during in vitro RNA synthesis, in particular the region located between nucleotides 219 and 239.

L17 ANSWER 6 OF 9 MEDLINE on STN DUPLICATE 3
AB Human La autoantigen has been shown to influence internal initiation of translation of hepatitis C virus (HCV) RNA. Previously, we have demonstrated that, among the three RRMs of La protein, the RRM2 interacts with HCV internal ribosome entry site (IRES) around the GCAC motif near the initiator AUG present in the stem region of stem-loop IV (SL IV) (Pudi, R., Abhiman, S., Srinivasan, N., and Das S. (2003) J. Biol. Chemical 278, 12231-12240). Here, we have demonstrated that the mutations in the GCAC motif, which altered the binding to RRM2, had drastic effect on HCV IRES-mediated translation, both in vitro and in vivo. The results indicated that the primary sequence of the stem region of SL IV plays an important role in mediating internal initiation. Furthermore, we have shown that the mutations also altered the ability to bind to ribosomal protein S5 (p25), through which 40 S ribosomal subunit is known to contact the HCV IRES RNA. Interestingly, binding of La protein to SL IV region induced significant changes in the circular dichroism spectra of the HCV RNA indicating conformational alterations that might assist correct positioning of the initiation complex. Finally, the ribosome assembly analysis using sucrose gradient centrifugation implied that the mutations within SL IV of HCV IRES impair the formation of functional ribosomal complexes. These observations strongly support the hypothesis that La protein binding near the initiator AUG facilitates the interactions with ribosomal protein S5 and 48 S ribosomal assembly and influences the formation of functional initiation complex on the

HCV IRES RNA to mediate efficient internal initiation of translation.

L17 ANSWER 7 OF 9 MEDLINE on STN DUPLICATE 4
AB Translation of the hepatitis C virus (HCV) RNA is mediated by the interaction of ribosomes and cellular proteins with an internal ribosome entry site (IRES) located within the 5'-untranslated region (5'-UTR). We have investigated whether small RNA molecules corresponding to the different stem-loop (SL) domains of the HCV IRES, when introduced in trans, can bind to the cellular proteins and antagonize their binding to the viral IRES, thereby inhibiting HCV IRES-mediated translation. We have found that a RNA molecule corresponding to SL III could efficiently inhibit HCV IRES -mediated translation in a dose-dependent manner without affecting cap-dependent translation. The SL III RNA was found to bind to most of the cellular proteins which interacted with the HCV 5'-UTR. A smaller RNA corresponding to SL e+f of domain III also strongly and selectively inhibited HCV IRES -mediated translation. This RNA molecule interacted with the ribosomal S5 protein and prevented the recruitment of the 40S ribosomal subunit. This study reveals valuable insights into the role of the SL structures of the HCV IRES in mediating ribosome entry. Finally, these results provide a basis for developing anti-HCV therapy using small RNA molecules mimicking the SL structures of the 5'-UTR to specifically block viral RNA translation.

L17 ANSWER 8 OF 9 MEDLINE on STN DUPLICATE 5
AB The inability for the internal ribosomal entry site (IRES) of hepatitis C virus (HCV) to be readily studied in the context of viral replication has been circumvented by constructing chimeras such as with poliovirus (PV), in which translation of the genome polyprotein is under control of the HCV IRES. During our attempts to configure the PV/HCV chimera for our drug discovery efforts, we discovered that an adenine- (A) to-guanine (G) change at nt 350 in domain IV of the HCV IRES resulted in a nonviable phenotype. Similarly, a mengovirus (MV)/HCV chimera using the same configuration with a G at nt 350 (G-350) was found to be nonviable. In contrast, a bovine viral diarrhea virus (BVDV)/ HCV chimera remained viable with G-350 in the HCV IRES insert. Second-site, resuscitating mutations were identified from the G-350 PV/HCV and MV/HCV viruses after blind passaging. For both viruses, the resuscitating mutations involved destabilization of domain IV in the HCV IRES. The nonviability of G-350 in the picornavirus/HCV chimeric background might be linked to translation efficiency as indicated by analyses with dual reporter and PV/HCV replicon constructs.

L17 ANSWER 9 OF 9 MEDLINE on STN DUPLICATE 6
AB The genome of hepatitis C virus (HCV) is a single-stranded RNA of positive polarity that has a poly(U/C) tract followed by a highly conserved 98-nt sequence, termed the X region, in the 3' untranslated region (UTR). To investigate the effect of the 3'UTR on the HCV translation that depends on the internal ribosomal entry site (IRES), we prepared a deletion HCV RNA, MA delta, that lacked the RNA region from nt 1286 to 8785. A series of MA delta RNAs that differ in the primary structure of their 3'UTR, were generated and examined for their translation efficiencies in reticulocyte lysates. Deletion of the poly(U/C) tract and/or stem-loop structure (SL) 3 region of 3'X resulted in enhancement of the translation efficiency. Translation of MA delta RNA was inhibited by the addition of recombinant polypyrimidine tract-binding protein (PTB). A similar inhibition by PTB,

however, was observed when an RNA lacking the poly(U/C) tract or SL3 region was used. The inhibitory effect by PTB was not obvious for MA delta (1041) RNA composed of nt 1 to 1041 but MA delta (8928) RNA composed of nt 1 to 1285 and nt 8786 to 8928. These results suggest that the observed down-regulation of HCV translation by the 3'UTR is mediated by some host factor(s) other than PTB, and that a PTB site for inhibition resides in the coding sequence of nt 1042 to 8928 of MA delta RNA.

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=> d 3 4

L17 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2005:1027012 CAPLUS
DN 143:320967
TI Preparation of a small synthetic hepatitis C virus IRES RNA, its inhibition effect on HCV IRES-mediated translation and antiviral uses thereof
IN Ray, Partho Sarothi; Das, Saumitra
PA Indian Institute of Science, India
SO PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005087923	A1	20050922	WO 2005-IN78	20050311
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	EP 1723231	A1	20061122	EP 2005-732989	20050311
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	US 20080015159	A1	20080117	US 2006-598700	20060908
PRAI	IN 2004-CH224	A	20040312		
	WO 2005-IN78	W	20050311		

RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 4 OF 9 MEDLINE on STN DUPLICATE 2
AN 2005360978 MEDLINE
DN PubMed ID: 16014945
TI A peptide derived from RNA recognition motif 2 of human la protein binds to hepatitis C virus internal ribosome entry site, prevents ribosomal assembly, and inhibits internal initiation of translation.
AU Pudi Renuka; Ramamurthy Sudhamani S; Das Saumitra
CS Department of Microbiology and Cell Biology, Indian Institute of Science, Bangalore.
SO Journal of virology, (2005 Aug) Vol. 79, No. 15, pp. 9842-53.
Journal code: 0113724. ISSN: 0022-538X.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200508
ED Entered STN: 15 Jul 2005
Last Updated on STN: 19 Aug 2005
Entered Medline: 18 Aug 2005

=> d 6 7

L17 ANSWER 6 OF 9 MEDLINE on STN DUPLICATE 3
AN 2004343440 MEDLINE
DN PubMed ID: 15138264
TI La protein binding at the GCAC site near the initiator AUG facilitates the ribosomal assembly on the hepatitis C virus RNA to influence internal ribosome entry site-mediated translation.
AU Pudi Renuka; Srinivasan Prabhavathi; Das Saumitra
CS Department of Microbiology and Cell Biology, Indian Institute of Science, Sir C.V. Raman Avenue, Bangalore 560012, India.
SO The Journal of biological chemistry, (2004 Jul 16) Vol. 279, No. 29, pp. 29879-88. Electronic Publication: 2004-05-10.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200408
ED Entered STN: 13 Jul 2004
Last Updated on STN: 18 Aug 2004
Entered Medline: 17 Aug 2004

L17 ANSWER 7 OF 9 MEDLINE on STN DUPLICATE 4
AN 2004155885 MEDLINE
DN PubMed ID: 15020704
TI Inhibition of hepatitis C virus IRES-mediated translation by small RNAs analogous to stem-loop structures of the 5'-untranslated region.
AU Ray Partho Sarothi; Das Saumitra
CS Department of Microbiology and Cell Biology, Indian Institute of Science, Bangalore 560012, India.
SO Nucleic acids research, (2004) Vol. 32, No. 5, pp. 1678-87. Electronic Publication: 2004-03-12.
Journal code: 0411011. E-ISSN: 1362-4962.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200407
ED Entered STN: 31 Mar 2004
Last Updated on STN: 7 Jul 2004
Entered Medline: 6 Jul 2004

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FULL ESTIMATED COST	121.17	123.90

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CA SUBSCRIBER PRICE                                   -3.20      -3.20

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FULL ESTIMATED COST                                    0.42     124.32

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CA SUBSCRIBER PRICE                                    0.00      -3.20

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  revised 2008 MeSH terms.  See HELP RLOAD for details.

  This file contains CAS Registry Numbers for easy and accurate
  substance identification.

  See HELP RANGE before carrying out any RANGE search.

=> s ires efficiency
          1517 IRES
        154593 EFFICIENCY
L18          9 IRES EFFICIENCY
                 (IRES(W)EFFICIENCY)

=> s l1 and genomic
        103372 GENOMIC
L19         92 L1 AND GENOMIC

=> s l18 and genomic
        103372 GENOMIC
L20          3 L18 AND GENOMIC

=> s l20 and structural
        277828 STRUCTURAL
L21          1 L20 AND STRUCTURAL

=> d

L21  ANSWER 1 OF 1     MEDLINE on STN
AN   2003247798     MEDLINE
DN   PubMed ID: 12771425
TI   Hepatitis C virus IRES efficiency is unaffected by the
     genomic RNA 3'NTR even in the presence of viral structural
     or non-structural proteins.
AU   Imbert Isabelle; Dimitrova Maria; Kien Francois; Kieny Marie Paule;
     Schuster Catherine
CS   INSERM U544, Institut de Virologie, 3 rue Koeberle, 67000 Strasbourg,
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     France.
SO   The Journal of general virology, (2003 Jun) Vol. 84, No. Pt 6, pp.
     1549-57.
     Journal code: 0077340. ISSN: 0022-1317.
CY   England: United Kingdom
DT   Journal; Article; (JOURNAL ARTICLE)
     (RESEARCH SUPPORT, NON-U.S. GOV'T)
LA   English
FS   Priority Journals
EM   200306
ED   Entered STN: 29 May 2003
     Last Updated on STN: 1 Jul 2003
     Entered Medline: 30 Jun 2003


=> s domain iiid
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           163 IIID
L22          6 DOMAIN IIID
                 (DOMAIN(W)IIID)

=> s l22 and 40s
          1889 40S
L23          3 L22 AND 40S

=> s l23 and ires
          1517 IRES
L24          3 L23 AND IRES

=> s l24 and complete
        350334 COMPLETE
L25          0 L24 AND COMPLETE

=> s l24 and compete
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L26          1 L24 AND COMPETE

=> d

L26  ANSWER 1 OF 1     MEDLINE on STN
AN   2003021215     MEDLINE
DN   PubMed ID: 12527783
TI   Antisense oligonucleotides targeted to the domain IIId
     of the hepatitis C virus IRES compete with 40S
     ribosomal subunit binding and prevent in vitro translation.
AU   Tallet-Lopez Beatrice; Aldaz-Carroll Lydia; Chabas Sandrine; Dausse Eric;
     Staedel Cathy; Toulme Jean-Jacques
CS   INSERM U386, Universite Victor Segalen, 146 rue Leo-Saignat, 33076
     Bordeaux, France.
SO   Nucleic acids research, (2003 Jan 15) Vol. 31, No. 2, pp. 734-42.
     Journal code: 0411011. E-ISSN: 1362-4962.
CY   England: United Kingdom
DT   Journal; Article; (JOURNAL ARTICLE)
     (RESEARCH SUPPORT, NON-U.S. GOV'T)
LA   English
FS   Priority Journals
EM   200303
ED   Entered STN: 16 Jan 2003
     Last Updated on STN: 7 Mar 2003
     Entered Medline: 6 Mar 2003
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FULL ESTIMATED COST                                       0.18     126.40

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CA SUBSCRIBER PRICE                                       0.00      -3.20

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